PROCEEDINGS OF THE BRITISH CARDIAC SOCIETY

The THIRTY-THIRD ANNUAL GENERAL MEETING of the British Cardiac Society was held at Downing College, Cambridge, by kind permission of Sir Lionel Whitby and the Fellows, on Friday, April 9, 1954. The Chairman, LESLIE COLE, took the Chair at 9.30 a.m.; 125 members and 19 visitors were present.

PRIVATE BUSINESS

1. The minutes of the last Annual General Meeting, having been published in the Journal (**15**, 461, 1953) were taken as read and confirmed.
2. The balance sheet for 1953–54 was presented, having been audited and found correct by G. E. S. Ward and D. Evan Bedford. The credit balance on April 3, 1954 was £782 4*s.* 10*d.*
3. R. C. Brock and A. Morgan Jones were elected members of the Council in place of G. E. S. Ward and D. Evan Bedford (terms of office expired).
4. The following Associate Members were elected as *Ordinary Members.*

P. H. Davison	F. McKeown	W. Somerville
K. W. Donald	A. Rogen	G. Thomas
J. Gough	H. Scarborough	G. M. Wilson
L. K. Malley		

5. The following *Surgical Members* were elected.

N. R. Barrett (London)	A. Logan (Edinburgh)
W. P. Cleland (London)	W. A. Mackey (Glasgow)

6. The following *Associate Members* were elected for a further period of three years.

W. E. Clarke	J. P. D. Mounsey
J. G. Graham	J. Tulloch
S. Howarth	A. W. W. B. Woods
R. M. Marquis	

7. The following new *Associate Members* were elected and introduced to the President and the Chairman.

W. D. Brinton (Winchester)	R. Mulcahy (Dublin)
R. M. Fulton (Manchester)	M. F. Oliver (Edinburgh)
A. D. Gillanders (Edinburgh)	A. Schott (London)
C. B. Henderson (Newcastle)	D. S. S. Short (London)
R. F. Jarrett (Gloucester)	P. Stock (Stoke-on-Trent)
T. D. V. Lawrie (Glasgow)	

8. The Secretary announced that arrangements had been made for thirty members of the British Cardiac Society to attend as delegates the Second International Congress of Cardiology to be held in Washington in September, 1954.
9. It was agreed that the original decisions of the Council under which the *British Heart Journal* had been conducted should be added as a new rule of the Society:

   " *British Heart Journal.* The Editors, and the Editorial Board, shall be appointed by the Council of the British Cardiac Society. The Editor of the *British Medical Journal* shall be a member of the Editorial Board. The Editors shall be appointed for a period not exceeding five years, and shall be eligible for re-appointment. The Editor or one of the Editors delegated by him to act as his deputy shall, *ex-officio,* be a member of the Council."
10. The question of wearing dinner-jackets for the dinner of the Annual General Meeting was discussed at length, and it was finally agreed that they should not be worn on every occasion, but only at the discretion of the Council.

SHORT COMMUNICATIONS

THE PATHOLOGICAL LESION IN ENDO-MYOCARDIAL FIBROSIS

By J. D. BALL and J. N. P. DAVIES (*introduced by J. McMichael*). The description is based on a study of 32 hearts from Uganda Africans dying in Mulago Hospital, Kampala.

The main lesions present are in the ventricles and the auriculo-ventricular valves. The left ventricle shows a thickened endocardium which is white and shiny. It may cover 60–90 per cent of the endocardial surface, and when extensive it commonly has a layer of mural thrombus on top of it. The fibrosis spreads from the apex up the posterior wall and commonly involves the posterior mitral cusp, which may become completely adherent to the ventricle. Such cases in life show the signs of mitral incompetence.

The right ventricle shows a similar lesion except for the fact that obliteration of the inflow tract always occurs if the lesion has developed to cover more than 30 per cent of the endocardium. The endocardial fibrosis involves the tricuspid valve in most cases, but the damage is much less than in the mitral valve. If the right ventricular lesions predominate, the case shows pure right heart failure, with or without the signs of tricuspid incompetence.

The common auricular lesion is mural thrombosis in the auricular appendage, particularly the right. Small fibrotic plaques are sometimes seen on the auricular wall near the mural thrombus.

Histological studies show a grossly thickened endocardium, acellular except in its deeper layers, where a belt of granulation tissue with dilated blood vessels is seen. The fibrous tissue may penetrate deeply into the myocardium, fibres of which normally show degenerative changes.

THE SICKLE-SHAPED R–T PLATEAU, A COMMON RS–T PATTERN IN HEALTH

By D. EVAN BEDFORD and GERALD THOMAS. The sickle-shaped R–T plateau comprises an elevated R–T segment with an upward concavity, terminating in a high positive and sharply-peaked T wave. The lowest point of the dipped R–T segment is nearer to R than to the summit of T. Typically, the QRS complex is predominantly positive, there is often a well marked Q wave, and the downstroke of R is notched, slurred, or may show an R^1.

The sickle-shaped R–T plateau shows mainly in either lead I or lead III, and also in the left chest leads. Reciprocal R–T displacement, as seen in cardiac infarction, never occurs.

The R–T plateau of pericarditis may be sickle-shaped or dome-shaped. When sickle-shaped, it may be indistinguishable from the R–T anomaly we have described, in an individual lead, but its distribution in the various leads is different, and it is, of course, a transient pattern.

The sickle-shaped R–T plateau appears to be a non-specific pattern, occurring in health and occasionally in heart disease. It may occur in cases of coronary disease. We have noticed it especially in cases of effort syndrome, liable to fainting. It has been described as occurring in athletes and in vagotonia.

VARIATIONS IN THE ELECTROCARDIOGRAM, PARTICULARLY IN THE T WAVES, ON STANDING

By A. J. V. CAMERON. The effect on the electrocardiogram of alteration of posture from lying to standing has been studied in forty healthy adults. On standing, the heart rate becomes faster and noteworthy changes may be seen in the P wave, QRS complex, and T wave.

Increased positivity of the P waves in leads II and III is common and is associated with decreased positivity in aVR and aVL and increased positivity in aVF and sometimes in V4 to V6.

The changes in the QRS complex can largely be explained by clockwise rotation of the heart around its antero-posterior and long axes and this usually causes a shift of the electrical axis to the right in the standard limb leads.

The T wave becomes either less positive in all three standard limb leads or less positive in leads II and III only. These appearances are due to increased positivity of the T wave in aVR, decreased positivity in aVF, and either increased positivity or no change in aVL. The change in aVL is never greater than that in aVR. In the præcordial leads, there is a general tendency to decrease in positivity of the T waves especially in V4 to V6, while there may be increased positivity in the right pectoral leads and occasionally also in V1 and V2. Often the polarity of the T waves is altered, for example upright T waves in leads II, III, aVF, and V4 to V6 may become flat, diphasic or inverted, while inverted T waves in aVR, aVL, and right pectoral leads may become flat, diphasic or upright. The most obvious effects are seen usually in the younger subjects.

These changes in the electrocardiogram occur immediately on standing and are at once reversed on lying down, though they may persist for as long as one hour with the subject standing at ease. Deep

inspiration sometimes enhances the changes and full expiration may lessen them. Prostigmin can prevent the changes occurring in the standing posture while atropine can produce them in the lying posture. Possible causal factors are discussed.

The Mitral Valve Pressure Gradient in Mitral Stenosis

By A. Venner (*introduced by H. E. Holling*). The pressure gradient across the mitral valve is measured by recording simultaneous left atrial and left ventricular pressures at operation. The study is facilitated by redrawing and superimposing the tracings. The left ventricular diastolic pressure shows considerable variance, and may rise or fall after valvotomy. For this reason the pressure gradient gives a better index, than left atrial pressure alone, of the severity of the obstruction from mitral stenosis, and of its relief by valvotomy. It is of practical value in operations on patients with both aortic and mitral stenosis in indicating the relative importance of the two lesions.

In normal rhythm there is a considerable increase in the pressure gradient (and presumably increased ventricular filling) coincident with atrial systole. In atrial fibrillation a higher mean atrial pressure is necessary to achieve the same ventricular filling. These observations offer an explanation of the increased disability that accompanies atrial fibrillation in mitral stenosis, even when controlled with digitalis.

In patients with mitral stenosis alone, the left ventricular diastolic pressure shows little increase as filling proceeds. In patients with aortic valve disease also, there is usually a steady rise amounting to 5 mm. Hg in all. This difference is presumably related to aortic reflex, and decreased distensibility of the ventricle due to hypertrophy, and increased residual blood in the ventricle at the end of systole.

Baroceptor Responses to Acute Rhythm Change

By Sheila Howarth and E. P. Sharpey-Schafer. Acute decrease in pulse pressure from a fast abnormal rhythm was followed by peripheral constriction while an increase in pulse pressure caused vasodilatation. Even a single large beat, such as that following an ectopic beat, produced detectable changes. The results are compatible with the theory of baroceptor response to rapid transients.

The Diagnosis of Tricuspid Stenosis

By R. V. Gibson (*introduced*) and Paul Wood. Twelve cases of mitral valve disease with tricuspid stenosis are presented, nine of which occurred in a consecutive series of 300 cases of mitral valve disease studied in detail by one of us (P.W.), an incidence of 3 per cent.

The clinical diagnosis of tricuspid stenosis was based on the following findings.

(1) A giant *a* wave in the jugular venous pulse in the absence of pulmonary hypertension.

(2) Considerable elevation of the jugular venous pressure in cases with auricular fibrillation, provided the ventricular rate was controlled and there were no signs of pulmonary hypertension.

(3) Absence of a deep *y* descent in the jugular pulse despite a very high venous pressure.

(4) A diastolic murmur heard over the tricuspid area, maximal on inspiration.

(5) A tall right atrial P wave in the absence of electrocardiographic evidence of considerable right ventricular hypertrophy.

(6) Conspicuous dilatation of the right atrium without enlargement of the pulmonary artery.

Eleven of these twelve cases were investigated by means of cardiac catheterization. A diastolic or presystolic pressure gradient across the tricuspid valve was demonstrated clearly in each instance. Although tricuspid stenosis modified the clinical features of mitral stenosis in all these eleven cases, the mitral lesion was more important in all but one. Tricuspid stenosis was clinically dominant, however, in the case that was not investigated physiologically.

Of the twelve cases nine were initially diagnosed at the bed-side and three were discovered during cardiac catheterization.

The Diagnosis of Mitral Incompetence by means of Cardiac Catheterization

By S. G. Owen (*introduced*) and Paul Wood. Wedged pulmonary artery pressure records from cases of mitral incompetence judged to be pure, dominant, or severe have been compared with those in pure mitral stenosis where that diagnosis has been adequately established. The time taken for inscription of the " y " descent was significantly longer in the stenosis group. The most stable expression of this proved to be the quotient of the calculated rate of fall (mm. Hg per second) divided by the absolute height of the preceding " v " wave (mm. Hg). The value so obtained was independent of individual fluctuations in

mean pressure and diastolic length and yielded a highly significant difference between the two groups. The results of the study suggest that the derivation of this ratio will be of assistance in evaluating pulmonary capillary venous pressure records in mitral valve disease.

The Murmurs of Tricuspid Incompetence

By O. Müller (Bergen, *introduced*) and J. Shillingford. Published in full: *Brit. Heart J.*, **16**, 195, 1954.

Isolated Pulmonary Stenosis: A Review of 29 Cases

By D. W. Barritt (*introduced by C. Bruce Perry*). Twenty-nine patients with isolated pulmonary stenosis have been investigated clinically, radiologically, and electrocardiographically: the majority were entirely free of symptoms but two were handicapped by dyspnœa and one by anginal pain.

Twenty-four of the 29 have been followed for from 5 to 21 years. Symptomatic deterioration was evident in two; there were progressive changes in the electrocardiogram, increasing degrees of right ventricular dominance, in five only. No deaths have occurred.

A pulmonary systolic thrill and murmur were constant. In more than half the cases the second heart sound was single. The most constant radiological feature was prominence of the main pulmonary artery segment. The electrocardiograms varied from normal to the most severe degree of right ventricular dominance. Cardiac catheterization was carried out in seven.

On clinical and electrocardiographic grounds five cases were considered to have extreme right ventricular hypertrophy. The three highest right ventricular pressures were found in this group. Two of the five were the only ones in the series with marked symptoms.

Inferior Vena Cava Opening into Left Atrium

By L. B. Cole and D. L. Gardner (*introduced*). The patient, a woman of 33, was cyanosed from the age of 4 years. She led a normal life at school and as a domestic servant, and married at the age of 19. Her only symptom was slight breathlessness on exertion and her only physical signs were cyanosis and clubbing of the fingers. Cardioscopy was normal and the cardiogram showed inverted T waves. She had two living children, one stillbirth, and three miscarriages. Her later years were complicated by toxæmia of pregnancy, multiple emboli, and suspected bacterial endocarditis from all of which she recovered, and she was leading a comparatively active life when she died suddenly.

At necropsy the hypertrophied and dilated vena cava, normal below the diaphragm and receiving the dilated hepatic veins, was found to drain into the left atrium. The right atrium received a normal coronary sinus and the superior vena cava. The latter was joined by the dilated azygos vein, and by a left innominate vein occluded by fibrous tissue. The intact atrial septum bore a normal fossa ovale. The ventricular septum was complete. The large pulmonary artery and the small aorta were normal in position. The ductus arteriosus was occluded. The posterior wall of the hypertrophied left ventricle contained an area of replacement fibrosis, and the right ventricle was small.

Valvotomy for Aortic Stenosis

By R. C. Brock. Experience in the surgical treatment of twenty cases of valvotomy for aortic stenosis is presented. The mortality and morbidity are discussed.

The importance of studying the pressure gradient across the valve, when the heart is exposed at operation, is emphasized.

Congenital Aortic Stenosis and its Surgical Treatment

By R. M. Marquis and Andrew Logan. Twenty-three cases of congenital aortic stenosis are reviewed in an attempt to ascertain the place of surgery in treatment. Their ages range from birth to 24 years, and their progress has been followed for up to 10 years. In each case heart disease was recognized in early childhood.

Five patients died as a result of the stenosis, one during investigation prior to surgery. Six were submitted to aortic valvotomy; subaortic obstruction had not been revealed on angiocardiography and was not found at thoracotomy. The operative approach was through the left ventricle, and the valve was ruptured by an expanding dilator without prior incision. There were no deaths. Benefit from the

operation was revealed by striking decrease in physical signs and by a change in the form of the arterial pressure wave. Four of the patients received immediate benefit, but significant aortic incompetence with cardiac enlargement was produced in the two others.

It is suggested that electrocardiographic evidence of marked left ventricular hypertrophy in growing children is indicative of severe stenosis, and that valvotomy should be performed in such patients before the development of cardiac enlargement or significant symptoms. A history of cardiac syncope is an urgent indication for operation.

Ostium Primum, Ostium Secundum, and Foramen Ovale

By R. Hudson. Proper development of the left ventricle in utero depends on its receiving blood from the right atrium, and nature ensures this by separating the atria by septa which are never complete. Even after birth there is a valvular opening which may persist in the adult. Two septa are concerned, the septum primum in which arise the ostium primum (which closes) and the ostium secundum (which persists), and the septum secundum which surrounds the foramen ovale (which also persists). The septa fuse, usually completely, so separating the atria. The margins of the foramen ovale are identified in the right atrium as the rim of the fossa ovalis, which is floored by the septum primum.

Excluding the very rare closure of the foramen ovale itself (making the septum secundum imperforate and causing atresia of the left ventricle), the following classification is based on the variations due to persistence, disposition, and size of the various ostia and the completeness of fusion of the septa. The terminology used brings it into line with the nomenclature of modern embryology.

Normal. Foramen ovale (septa overlapping ostia).

(1) Sealed or (2) Valvular.

Abnormal. Atrial Septal Defect (septa not overlapping ostia completely).

(1) Ostium secundum (usual type).

(2) Ostium primum (uncommon). (*a*) Alone, (*b*) with ostium secundum, or (*c*) with valvular foramen ovale.

This communication was accompanied by a demonstration of models of the septa and ostia, showing their formation and defects.

The Action of Hexamethonium Bromide on the Pulmonary Circulation in Mitral Stenosis

By L. G. Davies, Brenda Van Leuven (*introduced*) and J. P. Goodwin. Twelve patients with mitral stenosis and varying degrees of pulmonary arterial hypertension were investigated. The pulmonary arterial pressure was measured by cardiac catheterization, and the cardiac output by the Fick principle, in the recumbent position. After the patient had reached a steady state, 15–20 mg. of hexamethonium bromide was injected down the cardiac catheter, and the pulmonary arterial pressure measured at intervals, for 20 minutes, at the end of which the cardiac output was again estimated. The cardiac rate and the arterial blood pressure were also recorded before and after the administration of the drug. The dose used was determined by previous test doses in each patient, the smallest amount that produced a fall in the arterial blood pressure of not more than 10 mm. Hg in the recumbent position being given for the catheterization observations.

The results showed that in the patients with severe active pulmonary hypertension, hexamethonium produced a fall in the pulmonary arterial pressure of approximately 30 per cent. The reduction in pressure commenced within 2 minutes of the injection, was maximal at 10 minutes, and did not last more than 20 minutes. In patients with moderate pulmonary hypertension, the fall in pulmonary arterial pressure was less, while it was negligible in those with normal or slightly elevated pressures. In none of the cases showing an appreciable fall in pulmonary arterial pressure was there a significant alteration in cardiac output or heart rate. The fall in systemic blood pressure was slight or negligible in all cases. In two additional patients the "pulmonary capillary" pressure was measured before and after hexamethonium, and a reduction occurred. No untoward effects from hexamethonium were noted.

It is concluded that hexamethonium lowers the total pulmonary vascular resistance in patients with active pulmonary hypertension. This reduction is likely to be due to a fall in arteriolar resistance resulting from dilatation of the pulmonary arterioles mediated through the autonomic nervous system. These findings provide further evidence of functional constriction of the pulmonary arterioles, and the results are discussed in the light of the concept of a "protective" increase in arteriolar resistance, and of the possible treatment of pulmonary hypertension with hexamethonium or similar compounds.

The Diuretic Effects of Diamox, a Carbonic Anhydrase Inhibitor

By T. Hanley (*introduced by C. E. Davies*). A new diuretic (diamox or 6063) which acts by inhibiting the enzyme carbonic anhydrase in the renal tubules has recently been introduced. The effects of this substance have been studied in normal persons and in patients with heart failure, with particular reference to the effect on urinary excretion of sodium and water. There is little difference between the effects produced in these two groups. The diuretic action is transient and adaptation to the drug occurs within three days.

The normal function of the enzyme in tubular reabsorption is discussed and an account is given of the value and practical limitations of diamox as a diuretic in the treatment of heart failure.

Mitral Incompetence Resulting from Auricular Fibrillation

By Raymond Daley and I. K. R. MacMillan (*introduced*). In patients believed to be suffering from mitral stenosis who have auricular fibrillation, the left auricular pressure wave may closely resemble that obtained in patients suffering from mitral incompetence. In order to assess the significance of this, auricular fibrillation was produced electrically in a series of dogs. The left auricular pulse wave resembled that obtained from patients with fibrillation, and from dogs in which experimental mitral incompetence was produced. During auricular fibrillation Evan's blue injected into the left ventricle was aspirated from the left auricle before recirculation could have occurred. This did not occur in normal rhythm. Hence, in the dog, auricular fibrillation is accompanied by mitral incompetence.